SOGC CLINICAL PRACTICE GUIDELINES

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HEMORRHAGIC SHOCK

This document has been reviewed by the Clinical Practice Obstetrics Committee and approved by Executive and Council of the Society of Obstetricians and Gynaecologists of Canada.

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Abstract

Objective: To review the clinical aspects of hemorrhagic shock and provide recommendations for therapy.

Options: Early recognition of hemorrhagic shock and prompt systematic intervention will help avoid poor outcomes.

Outcomes: Establish guidelines to assist in early recognition of hemorrhagic shock and to conduct resuscitation in an organized and evidence-based manner.

Evidence: Medline references were sought using the MeSH term "hemorrhagic shock." All articles published in the disciplines of obstetrics and gynaecology, surgery, trauma, critical care, anesthesia, pharmacology, and hemotology between 1 January 1990 and 31 August 2000 were reviewed, as well as core textbooks from these fields. Selected references from these articles and book chapters were also obtained and reviewed. The level of evidence has been determined using the criteria described by the Canadian Task Force on the Periodic Health Examination.

Recommendations:

1. Clinicians should be familiar with the clinical signs of hemorrhagic shock. (III-B)
2. Clinicians should be familiar with the stages of hemorrhagic shock. (III-B)
3. Clinicians should assess each woman's risk for hemorrhagic shock and prepare for the procedure accordingly. (III-B)
4. Resuscitation from hemorrhagic shock should include adequate oxygenation. (II-3A)
5. Resuscitation from hemorrhagic shock should include restoration of circulating volume by placement of two large-bore IVs, and rapid infusion of a balanced crystalloid solution. (I-A)
6. Isotonic crystalloid or colloid solutions can be used for volume replacement in hemorrhagic shock (I-B). There is no place for hypotonic dextrose solutions in the management of hemorrhagic shock (I-E).
7. Blood component transfusion is indicated when deficiencies have been documented by clinical assessment or hematological investigations (II-2B). They should be warmed and infused through filtered lines with normal saline, free of additives and drugs (II-3B).
8. Vasoactive agents are rarely indicated in the management of hemorrhagic shock and should be considered only when volume replacement is complete, hemorrhage is arrested, and hypotension continues. They should be administered in a critical care setting with the assistance of a multidisciplinary team. (III-B)
9. Appropriate resuscitation requires ongoing evaluation of response to therapy, including clinical evaluation, and hematological, biochemical, and metabolic assessments. (III-B)
10. In hemorrhagic shock, prompt recognition and arrest of the source of hemorrhage, while implementing resuscitative measures, is recommended. (III-B)

Validation: These guidelines have been reviewed by the Clinical Practice Obstetrics Committee and approved by Executive and Council of the Society of Obstetricians and Gynaecologists of Canada.

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FOR INFORMATION ON THE SELF-DIRECTED LEARNING EXERCISE SEE PAGE 521.

INTRODUCTION

Hemorrhagic shock is a rare but serious complication, which may occur in many obstetrical or gynaecological situations. Hemorrhage is a leading cause of maternal death in the developing world.[1] Death and morbidity secondary to hemorrhage are becoming less common due to early recognition and intervention and improved availability of medical resources.[1] Ten recommendations for the management of hemorrhagic shock are listed in the following text and have been graded according to their level of evidence as determined by the criteria of the Canadian Task Force on the Periodic Health Examination (Table 1).[2]

HEMORRHAGIC SHOCK IN OBSTETRICS

Obstetrical hemorrhage is often acute, dramatic, and underestimated.[3] Postpartum hemorrhage is a significant cause of maternal death.[3] Management of postpartum hemorrhage has been reviewed in detail in SOGC *Clinical Practice Guidelines for the Prevention and Management of Postpartum Hemorrhage*.[3]

HEMORRHAGIC SHOCK IN GYNAECOLOGY

A surgical procedure is the most common antecedent of acute gynaecological hemorrhage, although patients will occasionally present with acute hemorrhage from a ruptured ectopic pregnancy or from a neoplasm.[1] Risk identification is important in counselling patients prior to surgery and in preparation of the surgical team. Any process that distorts pelvic anatomy, such as endometriosis, neoplasm, or adhesions, or that leads to an inflammatory response may be associated with increased intraoperative blood loss. Identification, isolation, and rapid control of bleeding encountered during the procedure will limit the total loss. The anatomy of the pelvis and landmarks of the vascular tree must be familiar to every pelvic surgeon.

Patients with delayed postoperative hemorrhage may present with bleeding from the wound or vagina or with evidence of a hemoperitoneum. Careful examination and resuscitation with definitive and prompt control of blood loss is required, which may require a return to the operating theatre.

CLINICAL PRESENTATION AND COMPLICATIONS OF HEMORRHAGIC SHOCK

Hemorrhage occurs when there is excessive external or internal blood loss.[4] A defined volume is difficult to measure in most situations, and the loss evaluated visually is often underestimated.[4] Shock occurs when there is hypoperfusion of vital organs. Hypoperfusion may be due to malfunction of the myocardium (cardiogenic shock), overwhelming infection leading to redistribution of circulating volume into the extravascular space (septic shock), or hypovolemia due to severe dehydration or hemorrhage (hypovolemic shock).[1] Signs and symptoms of hemorrhagic shock will vary depending on the volume and rate of blood loss (Table 2).[1]

The key systems affected by hemorrhagic shock are the central nervous, cardiac, and renal systems.[5] The central nervous system is able to function despite hypoperfusion, until the mean

TABLE 1

QUALITY OF EVIDENCE ASSESSMENT[2]	CLASSIFICATION OF RECOMMENDATIONS
The quality of evidence reported in these guidelines has been described using the Evaluation of Evidence criteria outlined in the Report of the Canadian Task Force on the Periodic Health Exam. I: Evidence obtained from at least one properly randomized controlled trial. II-1: Evidence from well-designed controlled trials without randomization. II-2: Evidence from well-designed cohort (prospective or retrospective) or case-control studies, preferably from more than one centre or research group. II-3: Evidence obtained from comparisons between times or places with or without the intervention. Dramatic results in uncontrolled experiments (such as the results of treatment with penicillin in the 1940s) could also be included in this category. III: Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.	Recommendations included in these guidelines have been adapted from the ranking method described in the Classification of Recommendations found in the Report of the Canadian Task Force on the Periodic Health Exam. A. There is good evidence to support the recommendation that the condition be specifically considered in a periodic health examination. B. There is fair evidence to support the recommendation that the condition be specifically considered in a periodic health examination. C. There is poor evidence regarding the inclusion or exclusion of the condition in a periodic health examination, but recommendations may be made on other grounds. D. There is fair evidence to support the recommendation that the condition not be considered in a periodic health examination. E. There is good evidence to support the recommendation that the condition be excluded from consideration in a periodic health examination.

TABLE 2
CLINICAL FEATURES OF HEMORRHAGIC SHOCK[1]

System	Early shock	Late shock
CNS	Altered mental status	Obtunded
Cardiac	Tachycardia	Cardiac failure
	Orthostatic hypotension	Arrhythmias
		Hypotension
Renal	Oliguria	Anuria
Respiratory	Tachypnea	Tachypnea
		Respiratory failure
Hepatic	No change	Liver failure
Gastrointestinal	No change	Mucosal bleeding
Hematological	Anemia	Coagulopathy
Metabolic	None	Acidosis
		Hypocalcemia
		Hypomagnesemia

arterial pressure falls below 60–70 mmHg.[1] With increasing severity of hypovolemia, mild agitation and confusion progress to lethargy and obtundation.[1] The heart plays an important role in the compensation for losses in early shock.[3] Early hypovolemia is associated with reflex tachycardia and increased stroke volume.[4,6] With continued loss, hypoperfusion of the coronary arteries and myocardium leads to cardiac dysfunction, ischemia, and failure;[1] symptoms of chest pain and dyspnea with signs of rales, tachypnea, and murmurs or arrhythmias are indicative of this process. The kidney will compensate for losses by activation of the renin-angiotensin-aldosterone system.[4] Early, reversible renal injury is associated with low urine sodium concentration and high urine osmolality (>500 mOsm).[1] Oliguria is a reliable sign that these compensatory mechanisms have been overwhelmed.[1]

All organ systems are ultimately affected in shock. Respiratory, hepatic, and gastrointestinal systems can be affected early in the process since cardiac output is redirected to the most important organs: the heart, brain, and kidneys.[6,7] Manifestations of lung injury include: dyspnea, tachypnea, pulmonary infiltrates, and edema leading to decreased tissue compliance and hypoxia. Symptoms of adult respiratory distress syndrome (ARDS) include: intrapulmonary shunting, reduced pulmonary compliance, and low arterial pO_2 that often requires assisted mechanical ventilation.[6,8] Moderate elevations of bilirubin and alkaline phosphatase can be seen with ischemic hepatic injury.[1] Gastrointestinal ischemia manifests as bleeding of either frank blood or coffee ground hematemesis or hematochezia or with delayed abdominal pain secondary to gut ischemia.[9] Erosion of the intestinal mucosa allows bacteremia and subsequent sepsis.[10] Multi-system injury can lead to coagulopathy, and metabolic disturbances such as acidosis.[1]

RECOMMENDATION

1. Clinicians should be familiar with the clinical signs of hemorrhagic shock. (III-B)

PATHOPHYSIOLOGY OF HEMORRHAGIC SHOCK

In hemorrhagic shock, an acute reduction in blood volume leads to sympathetic compensation by peripheral vasoconstriction, tachycardia, and increased myocardial contractility, which in turn increases the myocardial demand for oxygen, to a level that cannot be maintained.[1] Simultaneously, tissue hypoperfusion from precapillary vasoconstriction leads to anaerobic metabolism and acidosis.[11] Tissue hypoxia, acidosis, and the release of various mediators lead to a systemic inflammatory response.[5,11]

Reperfusion injury occurs when oxygen radicals released during the acute phase are systemically circulated as whole body perfusion is restored.[4,11,12] Humoral and cellular inflammatory systems are also activated, and contribute to vascular and cellular injury.[12,13] Transmigration of microorganisms and endotoxins across weakened mucosal barriers contributes to systemic inflammatory response syndrome (SIRS) and multiple organ failure.[4,10,11]

CLASSIFICATION OF HEMORRHAGIC SHOCK

A classification of hemorrhagic shock is outlined in Table 3. This type of classification may aid in determining the volume required for initial replacement, and the listed signs of shock in determining the severity of occult losses. The symptoms and sequelae of hemorrhage are ultimately related to perfusion of tissues. Loss of less than, or equal to, 15% of blood volume (compensated shock) may not be associated with any change in blood pressure (BP), pulse, or capillary refill. Mild shock is usually easily compensated, especially in the younger, healthy woman of reproductive age.[14] Further losses lead to tachycardia, a catecholamine response characterized by increased sympathetic tone. Resting BP is usually normal, but orthostatic changes in BP and pulse may be evident. Simple resuscitative measures will successfully reverse these changes.[1] Ongoing losses of blood volume may overtake the heart's ability to compensate, and marked tachycardia is associated with a fall in BP, classified as moderate shock. With continued bleeding, hypoperfusion of tissues occurs, leading to anaerobic metabolism and acidosis, classified as severe shock. The patient demonstrates marked tachycardia and tachypnea with respiratory failure, becomes oliguric, and then anuric. Obtundation and loss of consciousness may also occur.[1] Cellular dysfunction, followed by cell death, leads to multiple organ failure, resulting in irreversible shock.[1,15] The mortality rate at this stage is in excess of 30%.[1]

TABLE 3

CLASSIFICATION OF HEMORRHAGIC SHOCK[1,5,16]

	Compensated	Mild	Moderate	Severe
Blood Loss (mL)	≤1000	1000–1500	1500–2000	>2000
Heart rate (bpm)	<100	>100	>120	>140
Blood pressure	Normal	Orthostatic change	Marked fall	Profound fall
Capillary refill	Normal	May be delayed	Usually delayed	Always delayed
Respiration	Normal	Mild increase	Moderate tachypnea	Marked tachypnea: respiratory collapse
Urinary output (mL/h)	>30	20–30	5–20	Anuria
Mental status	Normal or agitated	Agitated	Confused	Lethargic, obtunded

RECOMMENDATION

2. Clinicians should be familiar with the stages of hemorrhagic shock. (III-B)

RISK FACTORS

Evaluation of all patients presenting for obstetrical care or surgery should include a complete medical history. A personal or family history of coagulopathy, or personal use of anticoagulants, should be documented. A complete physical examination may reveal extensive bruising or petechiae. Investigations to assess coagulation status should be obtained in these situations and consultation from other disciplines considered.

All proposed procedures should be reviewed with the patient. The risk of complications including hemorrhage should be outlined and the discussion documented in the chart.[17] Certain clinical conditions and their surgical management are associated with an increased risk of hemorrhage, such as ectopic pregnancy, myomectomy, abruptio placenta, placenta previa, and malignant disease.[17] In some situations, it may be appropriate to counsel women about autologous blood transfusion or hemodilution techniques.[17,18] Jehovah's Witnesses may require special consideration.[19]

RECOMMENDATION

3. Clinicians should assess each woman's risk for hemorrhagic shock and prepare for the procedure accordingly. (III-B)

MANAGEMENT

Early resuscitation includes control of bleeding and restoration of circulating blood volume for oxygenation of tissues.[16] Techniques to minimize blood loss should be applied whenever possible. Exposure of the bleeding site, experienced assistance, and sound knowledge of pelvic anatomy, as well as a calm, systematic approach to vascular areas, will be useful in the prompt control of hemorrhage. As soon as the first signs of excessive blood loss and shock are evident, assistance from other members of the health care team, which may include an anesthetist, a second gynaecologist, a general surgeon, a vascular surgeon, a critical care specialist, a hematologist, and experienced nursing staff, should be considered when appropriate and if available. Laboratory and blood bank services should be informed and available for support. Since cell death due to hypoxic injury is the final common pathway in shock, all efforts should be directed at restoring tissue oxygenation as soon as possible. A useful mnemonic to achieve this goal is ORDER: Oxygenate, Restore circulating volume, Drug therapy, Evaluate response to therapy, Remedy underlying cause.[1,14] Outcome is dependent on early recognition and on immediate aggressive therapy, which relies on two basic principles: replace losses and arrest bleeding.

OXYGENATION

The initial step in any patient resuscitation is to secure an airway and provide adequate oxygenation.[16] In most surgical situations, an airway will already be in place, managed by the anesthetist. If regional anesthesia has been used, supplemental oxygen should be applied.[4] Consideration should be given to endotracheal intubation, if the patient is becoming disoriented or is tiring, and in an obtunded patient should be instituted immediately.[1] After extensive fluid resuscitation, edema of the trachea may make intubation difficult. Positive ventilatory pressures may be required in those patients with decreased pulmonary compliance.

RECOMMENDATION

4. Resuscitation from hemorrhagic shock should include adequate oxygenation. (II-3A)

RESTORE CIRCULATING VOLUME

Intravascular replacement of blood volume lost may be accomplished using crystalloid, colloid, or blood products. Initial

therapy should consist of 1–2L of intravenous (IV) crystalloid.[1,16] Intravenous access should be of large calibre (14–16 gauge) and in multiple sites to facilitate rapid volume infusion.[5,16] A central line may be considered, but it does not appear to provide any advantage over peripheral lines for rapid infusion of volume.[5,16] The time and skill required to establish a central line, and the risk of complications such as pneumothorax, should also be considered.[1,5] Central venous pressure measurements may be helpful for safe resuscitation, if there has been injury to the cardiovascular system or vascular injury in the lung, as the amount of fluid required to restore tissue perfusion may be difficult to estimate. These patients are at risk for pulmonary edema and lung injury, if excessive fluid replacement is given.[6,7] In monitoring patients with multiple organ failure, central venous pressure measurements may also be useful in resuscitation and monitoring.[16] Normal central venous pressure is 5 mmHg (range 0–8 mmHg).[14] Elevated pressures are seen in fluid overload, right ventricular failure, pulmonary embolus, cardiac tamponade, and severe tricuspid regurgitation. Low values are seen with shock from hypovolemia, sepsis, and anaphylaxis.[14]

RECOMMENDATION

5. Resuscitation from hemorrhagic shock should include restoration of circulating volume by placement of two large-bore IVs, and rapid infusion of a balanced crystalloid solution. (I-A)

Crystalloid Solutions

Crystalloid solutions are electrolyte solutions administered intravenously. Advantages of crystalloid solutions include availability, safety, and low cost.[5,6] The main disadvantage of using crystalloid solutions is their rapid movement from the intravascular to the extravascular space, leading to three or more times requirement for replacement,[1,4,20,21] and resulting in tissue edema.[22] Ringer's lactate is preferred over normal saline to avoid hyperchloremic acidosis associated with prolonged use of sodium solutions.[1,23,24] Hypertonic salt solutions are not generally recommended because of the risk of electrolyte disturbances.[1,6,20,25] There is no role for hypotonic dextrose solutions in the management of hypovolemic shock.[21]

Colloid Solutions

Colloid solutions contain molecules designed to stay within the intravascular compartment.[1,6,11,20-23] These products include albumin, hydroxyethyl starch, dextrans, and gelatins. They are more expensive and less available than crystalloids.[22,24] Each of these oncotic replacement products carries the risk of reaction.[1,20,23] Some will bind calcium or affect circulating immunoglobulins.[6,7,20,26] There is no convincing evidence that colloid solutions offer any advantage over crystalloid solutions in the replacement of volume in hemorrhagic shock.[1,4,5-7,20-22,24,27,28] A recent review of the use of albumin in the treatment of hypovolemia suggests that its use may increase the risk of death.[29] Crystalloids and colloids may be used together.[22]

RECOMMENDATION

6. Isotonic crystalloid or colloid solutions can be used for volume replacement in hemorrhagic shock (I-B). There is no place for hypotonic dextrose solutions in the management of hemorrhagic shock (I-E).

Transfusion

Many blood products are available to restore circulating volume, and replace coagulation factors and oxygen-carrying capacity (Table 4). Component therapy allows specific replacement to address specific needs. Hypovolemia is best corrected with crystalloid solution. In hemorrhagic shock, packed red blood cells (PRBC) are most commonly used to restore intravascular volume and oxygen-carrying capacity. The oxygen-carrying capacity of most healthy individuals will not be compromised until the hemoglobin concentration falls below 60–70 g/L.[1,7,20,30,31] There is no recommended "threshold hemoglobin."[7,30] Blood losses greater than 20–25% or cases of documented or suspected coagulopathy may require replacement of coagulation factors; coagulation studies are recommended after transfusion of 5 to 10 units of PRBCs.[14]

Platelet transfusions are indicated in situations of significant thrombocytopenia (platelet count less than 20,000 to 50,000 per mm^3) and continued hemorrhage.[31] Coagulation factor concentrates are available for identified deficiencies, and fresh frozen plasma can be administered in acute situations where the partial thromboplastin time and prothrombin time

TABLE 4

INDICATIONS FOR BLOOD COMPONENT THERAPY[14,20,30-35]

Component	Indication	Usual starting dose
Packed RBC	Replacement of oxygen-carrying capacity	2–4 Units IV
Platelets	Thrombocytopenia or thrombasthenia with bleeding	6–10 Units IV
Fresh frozen plasma	Documented coagulopathy	2–6 Units IV
Cryoprecipitate	Coagulopathy with low fibrinogen	10–20 Units IV

are elevated. There is no indication for prophylactic administration of platelets, plasma, or specific components in resuscitation of hemorrhagic shock.[20,30-33] Immediate complications of blood transfusions are increased when large volumes of blood product are infused.[30] All blood products should be cross-matched and administered through filtered lines with normal saline, free of additives or drugs.[16] Acid-base and electrolyte disturbances can be evident with large volume transfusions.[16] Blood products should be warmed to prevent hypothermia.[1,30,32]

RECOMMENDATION

7. Blood component transfusion is indicated when deficiencies have been documented by clinical assessment or hematological investigations (II-2B). They should be warmed and infused through filtered lines with normal saline, free of additives and drugs (II-3B).

DRUG THERAPY

Vasoactive Agents

After adequate volume replacement has been achieved, vasoactive agents, which include inotropes and vasopressors, may be considered but are not often required in hemorrhagic shock.[1] When required, inotropic agents are administered first, followed by vasopressors in refractory cases. There is a risk that these agents may cause a further limitation of perfusion and oxygenation of distal organs.[7,14] Ideally, these drugs should be administered in a critical care setting with the assistance of a multidisciplinary team.

Dopamine can stimulate the function of alpha- and $beta_1$-adrenergic receptors.[7] At low doses of 1–3 μg/kg/min dopamine, the dopamine receptors in the cerebral, renal, and mesenteric circulation are stimulated, resulting in vasodilatation and increased urinary output. At moderate doses (2–10 μg/kg/min), alpha- and $beta_1$-adrenergic receptors are also stimulated, increasing myocardial contractility and cardiac output, resulting in an increase in myocardial oxygen consumption. At higher doses (>10 μg/kg/min), the alpha-adrenergic receptors are stimulated, leading to vasoconstriction and increases in blood pressure.[1,7]

Dobutamine is a $beta_1$- and $beta_2$-adrenergic stimulator. $Beta_2$ stimulation leads to systemic vasodilatation and reduced afterload.[7] Dobutamine is associated with less pulmonary congestion and less tachycardia than dopamine.[7]

Phenylephrine, norepinephrine, and epinephrine are used in situations of refractory shock. Their principal effect is to increase blood pressure by increasing systemic vascular resistance. They also produce some degree of coronary vasoconstriction, which may exacerbate myocardial ischemia (Table 5).[7]

RECOMMENDATION

8. Vasoactive agents are rarely indicated in the management of hemorrhagic shock and should be considered only when volume replacement is complete, hemorrhage is arrested, and hypotension continues. They should be administered in a critical care setting with the assistance of a multidisciplinary team. (III-B)

Other Drug Therapy

Broad coverage antibiotic therapy should be instituted in cases of hemorrhagic shock, as there is a significant risk of sepsis.[1] Ischemic injury to the gut makes it vulnerable to transmucosal

TABLE 5

PHARMACOLOGICAL SUPPORT OF THE CARDIOVASCULAR SYSTEM[1,7]

Agent	Usual dose range	Effect
Inotropic agents		
Dopamine	1–3 μg/kg/min	Increased renal output Vasodilation
	2–10 μg/kg/min	Increased heart rate Increased cardiac output
	>10 μg/kg/min	Peripheral vasoconstriction Increased heart rate and contractility
Dobutamine	2–10 μg/kg/min	Increased heart rate and contractility Decreased afterload
Vasopressor agents		
Phenylephrine	1–5 μg/kg/min	Peripheral vasoconstriction
Norepinephrine	1–4 μg/min	Peripheral vasoconstriction
Epinephrine	1–8 μg/min	Peripheral vasoconstriction

erosions and may lead to bacteremia.[10] The gastric mucosa is susceptible to stress ulceration, which can be reduced by the use of antacids or H_2 blockers.[1]

Stroma-free hemoglobin (diaspirin cross-linked hemoglobin) is a new product currently under evaluation, which can replace the oxygen-carrying capacity of PRBCs. Its affinity for nitric oxide contributes to peripheral vasoconstriction. Potential advantages include a longer shelf life and universal compatibility.[35] There is no clear clinical advantage over PRBCs. Risks include extravasation, possible coagulopathy, and the risks associated with human blood components, as well as documented toxicities.[15,20,35-37]

EVALUATION OF RESPONSE TO THERAPY

Once oxygenation and circulating volume have been restored, re-evaluation of the clinical situation is in order. Vital signs, mental status, urinary output, and capillary refill should be assessed regularly throughout resuscitation.[14,16] Initiation of central monitoring may be indicated at this time, if the response to initial resuscitation has been less than expected or if blood loss is ongoing.[7] Blood should be drawn to assess hematological, coagulation, electrolyte, and metabolic status. Electrolyte and metabolic disorders as well as coagulation deficiencies should be corrected. Arterial blood gases should be obtained to determine adequacy of oxygenation. Management of alterations to oxygenation, ventilation, pH, fluid, and electrolyte balance should now be made based on laboratory measurements.[16] Blood components may also be used to replace identified deficiencies.

RECOMMENDATION

9. Appropriate resuscitation requires ongoing evaluation of response to therapy, including clinical evaluation, and hematological, biochemical, and metabolic assessments. (III-B)

REMEDY THE UNDERLYING CAUSE

Most cases of unresponsive shock in gynaecologic patients are due to ongoing losses of blood volume. While initial stabilization is taking place, attention should be directed to the prompt arrest of bleeding. Aggressive restoration of normal blood pressure without arrest of hemorrhage will enhance further losses of blood volume by increasing flow and impeding coagulation at the site of injury.[11,15,25,27,38-40] Mild to moderate hypotension allows clot formation and slows bleeding from the injured vessel.[39] The source of blood loss should be identified and arrested as soon as possible.[14] Occult retroperitoneal hemorrhage should be suspected in patients who have a deteriorating hemodynamic status more than 12 hours post surgery.[17] In these cases, identification of the exact site of bleeding can be very difficult. Hemorrhage during or following an operative procedure should be addressed surgically, with immediate exploration, isolation, and ligation of bleeding vessels. In most cases, hemostasis can be achieved in this manner. Isolation and ligation of the uterine or internal iliac arteries are useful techniques to control hemorrhage in the pelvis.[17] The ureter is especially vulnerable during the placement of hemostatic sutures in the pelvis. If hemorrhage continues, coagulation should be evaluated and any identified deficiencies should be corrected. Adjunctive measures such as radiologic embolization and packing of the pelvis should be considered in refractory cases.[17]

Careful documentation of events and the interventions performed should be completed in a timely fashion. Communication with the patient, her partner, and her family should be prompt, frequent, and clear.

RECOMMENDATION

10. In hemorrhagic shock, prompt recognition and arrest of the source of hemorrhage, while implementing resuscitative measures, is recommended. (III-B)

CONCLUSION

Hemorrhagic shock may occur in many obstetrical or gynaecological conditions. Prompt recognition of blood losses and implementation of resuscitative measures in a calm and systematic fashion are imperative. A multidisciplinary approach to the multisystem effects of shock is essential to optimize the outcome.

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